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EDITORIAL

Setting the ventilator in acute brain injury

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Studies of patients receiving invasive mechanical ventilation (IMV) consistently demonstrate improved clinical outcomes with the use of a lung-protective strategy [1–3]. However, uncertainty remains regarding the safety and efficacy of this strategy in patients with acute brain injury (ABI; defined here as patients with ischemic stroke, traumatic brain injury, or intra-cranial hemorrhage). Brain-injured patients have historically received higher tidal volume (Vt) and lower positive end expiratory pressure (PEEP) compared to other critically ill patients [4], partly due to concerns that lower Vt could increase intra-cranial pressure (ICP) (via hypercapnic acidosis and cerebrovascular vasodilation) [5], and that higher levels of PEEP could impair cerebral perfusion pressure (via reductions in cardiac venous return, cardiac output, and cerebral venous outflow) [5]. Notwithstanding, recent findings support the safety of a protective ventilation strategy in the majority of patients with ABI [5–7]. This strategy can be implemented in a range of ABI patients without substantial perturbations in arterial partial pressure of carbon dioxide ($PaCO_2$) and ICP, and with potential improvement in clinical outcomes [6]. Studies have also indicated that higher PEEP may ameliorate brain tissue oxygenation, provided that systemic hemodynamic status is maintained [8]. While robust evidence is lacking, protective ventilation is now recommended in patients with ABI [7]. Here, we outline a concise strategy for IMV which considers ventilation settings, arterial blood gas targets, and a specific approach in patients who have concurrent ABI and acute respiratory distress syndrome (ARDS).

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Ventilatory settings

When selecting IMV settings, a reasonable first step is to set Vt between 4 and 8 ml kg^{-1} of predicted body weight (PBW) [3], while ensuring that plateau pressure (Pplat) is ≤ 30 cmH_2O. Vt in this range is deemed safe in most patients with ABI. The selection of initial respiratory rate in controlled modes of IMV should be titrated to the first blood gas to maintain $PaCO_2$ between 35 mmHg and 45 mmHg, as discussed below [5, 6].

Regarding ventilator mode, common practice is to use volume-assist or pressure-assist control to precisely manage Vt and $PaCO_2$ in the early period of high acuity, and then to transition to pressure- or volume-support modalities when the patient is deemed ready. However, other IMV modes could be considered, and current guidelines do not make specific recommendations [7].

In general, the same level of PEEP should be used patients with ABI as in those without [7], although ABI patients should be closely monitored for undesired effects of PEEP on ICP and cerebral perfusion pressure. As PEEP increases intrathoracic pressure, this induces decreased venous-pressure return and consequently decreased cardiac output. This phenomenon is particularly explicit in the context of hypovolemia. If there is evidence of intravascular volume depletion, fluid loading is recommended prior to PEEP augmentation to mitigate indirect effects on cardiac output and cerebral perfusion pressure. If an increase in ICP is noted in conjunction with up-titration of PEEP, the latter should be decreased to maintain ICP within the targeted range.

Recent data suggest that patients with ABI could also benefit from approaches which minimize driving pressure and mechanical power [9, 10], or other individualized targeting in specific clinical scenarios (i.e., severe intra-cranial hemorrhage without ICP monitoring) but no solid evidence-based guidance can be formulated at this point, and prospective studies in this domain are ongoing (clinicaltrials.gov identifier NCT04459884).

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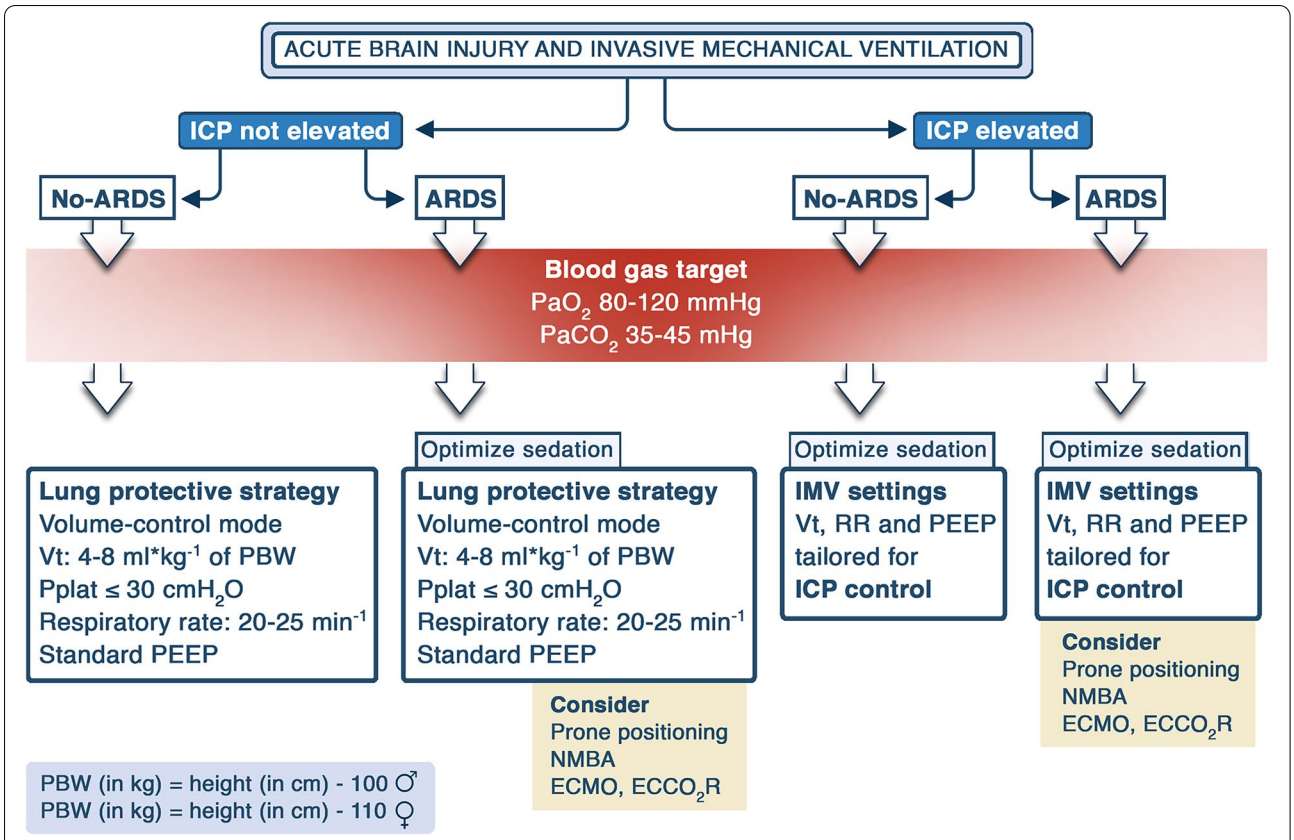

Fig. 1 Invasive mechanical ventilation in patients with acute brain injury. The first step is to set tidal volume within a range of 4–8 ml kg^{-1} of predicted body weight (a simplified formula is provided in the figure), using a volume-assist control mode. One possible option is to begin with 6 ml kg^{-1} PBW and further adjusting Vt to maintain Pplat ≤ 30 cmH_2O. A standard PEEP of at least 5 cmH_2O should be considered. The FiO_2 is set to achieve a PaO_2 of 80 mmHg to 120 mmHg. The target $PaCO_2$ should range between 35 and 45 mmHg. *ARDS* acute respiratory distress syndrome, *ECMO* extracorporeal membrane oxygenation, *$ECCO_2R$* extracorporeal carbon dioxide removal, *ICP* intra-cranial pressure, *IMV* invasive mechanical ventilation, *PBW* predicted body weight, *PEEP* positive end-expiratory pressure, *Pplat* plateau pressure, *NMBA* neuromuscular blocking agent, *RR* respiratory rate, *Vt* tidal volume

Oxygen and carbon dioxide targets

Given the need for continuous hemodynamic monitoring and repeated blood gas analysis, an arterial catheter should be considered in the early management of all severe ABI patients. Guidelines recommend targeting an arterial partial pressure of oxygen (PaO_2) of 80–120 mmHg in patients with ABI [7]. A recent study found that a PaO_2 outside the range of 92–156 mmHg was associated with higher in-hospital mortality [11], indicating that optimal PaO_2 targets are likely to evolve as more data becomes available. Strict control of $PaCO_2$ aims to prevent cerebral ischemia from hypocapnia-induced cerebrovascular constriction or from hypercapnia-induced cerebral vasodilation. A recent post-hoc analysis of 1476 patients with ABI found an association between severe hypocapnia or hypercapnia and increased in-hospital mortality [12]. Guidelines not only recommend a target $PaCO_2$ range of 35–45 mmHg, but also support temporary hyperventilation (hypocapnia) in the acute management of brain herniation and/or sudden ICP elevations [7]

Management of concurrent acute brain injury and acute respiratory distress syndrome

In patients with concurrent ABI and ARDS, a stepwise approach has been proposed which considers the relative severity of ABI and ARDS and integrates interactions between cerebral and cardio-thoracic physiology in all IMV decisions [13]. Lung-protective ventilation should be strongly considered; however evidence is lacking and a clear recommendation could not made in the latest consensus guideline [7]. Titration and optimization of sedation is critical to enable effective management of patients who have both ARDS and elevated ICP.

Management priorities in this subgroup are dynamic and clinician emphasis may need to shift between the brain and the lungs depending on dynamically evolving organ-specific physiology. Multimodal neuromonitoring should be considered, especially if the patient is comatose, requires deep sedation, or if higher levels of PEEP are necessary. In the presence of severe ARDS, adjunctive therapies such as prone positioning, use of neuromuscular blockers, or extracorporeal life support may be discussed on a case-by-case basis. Principles of IMV management in patients with ABI are summarized in the Fig. 1.

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Declarations

Conflict of interest
The authors do not have any conflict of interest to declare for the present work.

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